

# In the United States Court of Federal Claims

## OFFICE OF SPECIAL MASTERS

No. 17-1475V

Filed: October 12, 2022

PUBLISHED

KATHERINE KELLY,

Petitioner,

v.

SECRETARY OF HEALTH AND  
HUMAN SERVICES,

Respondent.

Special Master Horner

Entitlement; influenza (“flu”) vaccine; polymyalgia rheumatica (“PMR”); denial.

*Diana Stadelnikas, Maglio Christopher and Toale, Sarasota, FL, for petitioner.  
Kyle Pozza, U.S. Department of Justice, Washington, DC, for respondent.*

### **DECISION**<sup>1</sup>

On October 10, 2017, petitioner filed a petition under the National Childhood Vaccine Injury Act, 42 U.S.C. § 300aa-10-34 (2012),<sup>2</sup> alleging that the influenza (“flu”) vaccine she received on September 30, 2016, caused her to develop polymyalgia rheumatica (“PMR”). (ECF No. 1.) For the reasons set forth below, I conclude that petitioner is not entitled to compensation for her PMR.

#### **I. Applicable Statutory Scheme**

Under the National Vaccine Injury Compensation Program, compensation awards are made to individuals who have suffered injuries after receiving vaccines. In general, to gain an award, a petitioner must make a number of factual demonstrations, including showing that an individual received a vaccination covered by the statute;

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<sup>1</sup> Because this decision contains a reasoned explanation for the special master’s action in this case, it will be posted on the United States Court of Federal Claims’ website in accordance with the E-Government Act of 2002. See 44 U.S.C. § 3501 note (2012) (Federal Management and Promotion of Electronic Government Services). **This means the decision will be available to anyone with access to the Internet.** In accordance with Vaccine Rule 18(b), petitioner has 14 days to identify and move to redact medical or other information the disclosure of which would constitute an unwarranted invasion of privacy. If the special master, upon review, agrees that the identified material fits within this definition, it will be redacted from public access.

<sup>2</sup> Within this decision, all citations to § 300aa will be the relevant sections of the Vaccine Act at 42 U.S.C. § 300aa-10-34.

received it in the United States; suffered a serious, long-standing injury; and has received no previous award or settlement on account of the injury. Finally – and the key question in most cases under the Program – the petitioner must also establish a *causal link* between the vaccination and the injury. In some cases, the petitioner may simply demonstrate the occurrence of what has been called a “Table Injury.” That is, it may be shown that the vaccine recipient suffered an injury of the type enumerated in the “Vaccine Injury Table,” corresponding to the vaccination in question, within an applicable time period following the vaccination also specified in the Table. If so, the Table Injury is presumed to have been caused by the vaccination, and the petitioner is automatically entitled to compensation, unless it is affirmatively shown that the injury was caused by some factor other than the vaccination. § 300aa-13(a)(1)(A); § 300 aa-11(c)(1)(C)(i); § 300aa-14(a); § 300aa-13(a)(1)(B).

In many cases, however, the vaccine recipient may have suffered an injury *not* of the type covered in the Vaccine Injury Table. In such instances, an alternative means exists to demonstrate entitlement to a Program award. That is, the petitioner may gain an award by showing that the recipient’s injury was “caused-in-fact” by the vaccination in question. § 300aa-13(a)(1)(B); § 300aa-11(c)(1)(C)(ii). In such a situation the presumptions available under the Vaccine Injury Table are inoperative. The burden is on the petitioner to introduce evidence demonstrating that the vaccination actually caused the injury in question. *Althen v. Sec’y of Health & Human Servs.*, 418 F.3d 1274, 1278 (Fed. Cir. 2005); *Hines v. Sec’y of Health & Human Servs.*, 940 F.2d 1518, 1525 (Fed. Cir. 1991). Because PMR is not listed as an injury on the Vaccine Injury Table, petitioner must satisfy this burden of proof.

The showing of “causation-in-fact” must satisfy the “preponderance of the evidence” standard, the same standard ordinarily used in tort litigation. § 300aa-13(a)(1)(A); *see also Althen*, 418 F.3d at 1279; *Hines*, 940 F.2d at 1525. Under that standard, the petitioner must show that it is “more probable than not” that the vaccination was the cause of the injury. *Althen*, 418 F.3d at 1279. The petitioner need not show that the vaccination was the sole cause of the injury or condition, but must demonstrate that the vaccination was at least a “substantial factor” in causing the condition, and was a “but for” cause. *Shyface v. Sec’y of Health & Human Servs.*, 165 F.3d 1344, 1352 (Fed. Cir. 1999). Thus, the petitioner must supply “proof of a logical sequence of cause and effect showing that the vaccination was the reason for the injury;” the logical sequence must be supported by “reputable medical or scientific explanation, *i.e.*, evidence in the form of scientific studies or expert medical testimony.” *Althen*, 418 F.3d at 1278; *Grant v. Sec’y of Health & Human Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992). A petitioner may not receive a Vaccine Program award based solely on his or her assertions; rather, the petition must be supported by either medical records or by the opinion of a competent physician. § 300aa-13(a)(1).

In what has become the predominant framing of this burden of proof, the *Althen* court described the “causation-in-fact” standard, as follows:

Concisely stated, *Althen*'s burden is to show by preponderant evidence that the vaccination brought about her injury by providing: (1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of proximate temporal relationship between vaccination and injury. If *Althen* satisfies this burden, she is entitled to recover unless the [government] shows, also by a preponderance of the evidence, that the injury was in fact caused by factors unrelated to the vaccine.

*Althen*, 418 F.3d at 1278 (citations omitted). The *Althen* court noted that a petitioner need not necessarily supply evidence from medical literature supporting petitioner's causation contention, so long as the petitioner supplies the medical opinion of an expert. *Id.* at 1279-80. That expert's opinion must be based upon "sound and reliable" scientific explanation. *Boatmon v. Sec'y of Health & Human Servs.*, 941 F.3d 1351, 1359 (Fed. Cir. 2019) (quoting *Knudsen v. Sec'y of Health & Human Servs.*, 35 F.3d 543, 548-49 (Fed. Cir. 1994)). The *Althen* court also indicated that, in finding causation, a Program factfinder may rely upon "circumstantial evidence," which the court found to be consistent with the "system created by Congress, in which close calls regarding causation are resolved in favor of injured claimants." 418 F.3d at 1280.

## II. Procedural History

On October 10, 2017, petitioner filed her petition, alleging that the flu vaccine she received on September 30, 2016, caused her to develop PMR. (ECF No. 1.) This case was initially assigned to Special Master Millman. (ECF No. 6.) Petitioner subsequently filed medical records in support of her claim on October 10, 2017, and March 16, 2018, followed by a Statement of Completion on May 16, 2018. (ECF Nos. 7, 12, 14.) After reviewing petitioner's materials, respondent filed his Rule 4(c) report contesting entitlement on July 25, 2018. (ECF No. 19.) Respondent argued that petitioner failed to substantiate that her flu vaccine was the cause-in-fact of her PMR. (*Id.* at 5.)

In response to respondent's Rule 4(c) report recommending against compensation, petitioner filed a report from rheumatologist Thomas Zizic, M.D., on May 24, 2019. (ECF No. 28; Ex. 10.) The case was subsequently reassigned to my docket on June 4, 2019. (ECF No. 32.) On September 3, 2019, respondent filed a responsive report from immunologist and rheumatologist Mehrdad Matloubian, M.D., Ph.D. (ECF No. 34; Ex. A.) Petitioner then submitted a supplemental report from Dr. Zizic on May 6, 2020. (ECF No. 42; Ex. 40.) The expert report stage concluded with respondent's filing of an additional report from Dr. Matloubian on August 17, 2020. (ECF No. 44; Ex. C.)

On November 17, 2020, the parties indicated that the case was ripe for an entitlement determination. (ECF No. 47.) Petitioner proposed filing a motion for a ruling on the written record. (*Id.*) On May 3, 2021, petitioner filed her motion for a ruling on the written record and accompanying memorandum. (ECF No. 50.) Respondent filed

his responsive memorandum on September 29, 2021. (ECF No. 54.) Petitioner filed a reply on October 6, 2021. (ECF No. 55.)

Special masters “must determine that the record is comprehensive and fully developed before ruling on the record.” *Kreizenbeck v. Sec’y of Health & Human Servs.*, 945 F.3d 1362, 1366 (Fed. Cir. 2020); see also Vaccine Rule 8(d); Vaccine Rule 3(b)(2). The parties must have a full and fair opportunity to present their case and develop a record sufficient for review. *Id.* In light of all of the above, and upon review of the entire record, I conclude that petitioner has had a full and fair opportunity to develop the record of this case and that the case is ripe for resolution on the existing record.

### III. Factual History

#### a. As Reflected in the Medical Records

Petitioner’s pre-vaccination medical history included hyperkalemia and attention deficit hyperactivity disorder (“ADHD”), for which she took Adderall. (See, e.g., Ex. 3, pp. 6-7, 13, 17.) Before vaccination, petitioner exercised regularly and ran two marathons in 2013. (*Id.* at 17, 21.) Petitioner was seventy-three years old when she received the high dose flu vaccine on September 30, 2016. (Ex. 1, p. 1; see also Ex. 3, p. 7.)

On October 14, 2016, petitioner left a phone message for her primary care provider, Richard Bransdorf, M.D. (Ex. 4, p. 8.) She reported that she had a fever for two days following the flu shot and subsequently developed joint and bone pain in her shoulder, hips, and back. (*Id.*) A handwritten reply noted that petitioner’s complaints were “likely d/t [due to] shot,” and recommended that petitioner treat her symptoms with Tylenol or Aleve and schedule an appointment if they did not resolve within a few days.<sup>3</sup> (*Id.*) Petitioner left another phone message on October 17, 2016, reporting that her pain was gradually improving but remained persistent. (*Id.* at 6.) The physician’s assistant (“PA”), David Burns, noted that it “sounds like [a] flu like reaction to [the] vaccine” and stated that if petitioner was improving, there was no need for concern. (*Id.*)

Petitioner visited Dr. Bransdorf on November 18, 2016. (Ex. 3, pp. 6-7.) Dr. Bransdorf documented that petitioner received the flu vaccine on September 30, 2016, and later developed myalgias and fever. (*Id.* at 7.) Petitioner reported that her symptoms continued to wax and wane and that the pain was primarily in her shoulder and groin. (*Id.*) She noted that she was exhausted because her pain prevented her from resting and that she had difficulty lifting her arms above her head and pulling on a shirt. (*Id.*) During the visit, petitioner was afebrile and exhibited no neurological symptoms. (*Id.*) Upon examination, Dr. Bransdorf observed tenderness of the shoulders and groin bilaterally. (*Id.*) Dr. Bransdorf’s impression was myalgia/myositis. (*Id.*) Dr. Bransdorf prescribed 20 mg of prednisone and ordered laboratory tests. (*Id.*)

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<sup>3</sup> The signature below the handwritten note is illegible, and it is unclear who authored the note. (See Ex. 4, p. 8.) Petitioner asserts that Dr. Bransdorf is presumably the author of the handwritten note because he was petitioner’s covering physician. (See ECF No. 50, pp. 2, 17; ECF No. 55, p. 6.)

Petitioner's erythrocyte sedimentation rate ("ESR") was normal, but her C-reactive protein ("CRP"), an inflammatory marker, was elevated. (*Id.* at 35.)

On November 28, 2016, petitioner returned to Dr. Bransdorf for a follow-up visit. (Ex. 3, pp. 8-9.) Dr. Bransdorf noted that petitioner began prednisone due to possible PMR. (*Id.* at 9.) Petitioner reported that the prednisone provided "almost immediate relief." (*Id.*) Dr. Bransdorf's diagnosis at this visit was PMR. (*Id.*) He instructed petitioner to continue taking prednisone and tapered her dose to 10 mg per day. (*Id.*)

Petitioner returned to Dr. Bransdorf on January 10, 2017. (*Id.* at 10-11.) She reported that she remained on 10 mg of prednisone and was doing well. (*Id.* at 11.) However, she noted that she suffered a flare in symptoms after running five miles. (*Id.*) The plan was for her to continue 10 mg of prednisone for three months and limit her exercise. (*Id.*)

At a follow-up visit with Dr. Bransdorf on May 15, 2017, petitioner reported that she had been doing well but recently experienced neck pain. (Ex. 5, pp. 6-7.) Petitioner was having difficulty sleeping due to the neck pain. (*Id.* at 7.) Dr. Bransdorf noted that it was unclear whether petitioner's neck pain was due to a PMR flare or a mechanical injury. (*Id.*) At the time of the visit, she was taking 10 mg of prednisone daily. (*Id.*) A neck examination revealed tender paraspinal muscles with normal range of motion. (*Id.*) Dr. Bransdorf increased petitioner's prednisone dose to 20 mg daily, prescribed a muscle relaxer, and ordered an x-ray. (*Id.*) The cervical spine x-ray showed mild degenerative spondylosis and diffuse bony demineralization. (*Id.* at 14.)

Petitioner emailed Dr. Bransdorf on May 28, 2017, to clarify that she believed her neck pain began in late April after a long car ride. (*Id.* at 16.) She explained that it started in the right side of her neck and extended behind her ear and that it was tender when she pushed on it. (*Id.*) She stated that the pain then increased, progressed to her back, and radiated to her head and throat. (*Id.*) Petitioner also noted that the muscle relaxer did not help at first but provided some pain relief after multiple doses. (*Id.*) Given the headache she experienced, she was concerned for possible giant cell arteritis ("GCA"). (*Id.*)

During a visit with Dr. Bransdorf on August 8, 2017, petitioner reported that she had been taking 20 mg of prednisone, that her neck and shoulder pain had improved, and that she had some muscle aches. (Ex. 6, p. 7.) Petitioner's blood pressure was elevated, which was likely due to her steroid use. (*Id.*) Her physical examination was normal. (*Id.* at 8.) Dr. Bransdorf directed petitioner to taper the prednisone and begin anti-hypertensive medication. (*Id.*) A subsequent bone density study to screen for osteoporosis on August 17, 2017, showed petitioner's bone density had worsened since a previous study on July 25, 2011. (*Id.* at 11.)

On November 28, 2017, petitioner returned to Dr. Bransdorf. (Ex. 7, pp. 2-6.) Petitioner reported that she was taking 15 mg of prednisone and that she had arthralgia and joint pains but no muscle aches. (*Id.* at 5.) Her CRP was elevated, but her physical

examination was normal. (*Id.* at 4-5.) Dr. Bransdorf directed petitioner to remain on her medications. (*Id.* at 5.) Petitioner continued to follow up with Dr. Bransdorf routinely. (See, e.g., Ex. 8 pp. 11-14 (2/28/2018 visit); *id.* at 7-11 (8/9/2018 visit); *id.* at 4-7 (11/21/2018 visit).)

Petitioner returned to Dr. Bransdorf on February 21, 2019, to follow up on her current medications. (Ex. 42, p. 9.) Petitioner reported that she was doing well but still had shoulder and hip aches. (*Id.* at 11.) At the time of this visit, petitioner was on 5 mg of prednisone per day. (*Id.*) Dr. Bransdorf increased petitioner's prednisone dose to 10 mg per day with instructions to begin tapering after two weeks to help with her myalgias. (*Id.*)

On March 21, 2019, petitioner saw PA Burns for complaints of a vision disruption in her right eye. (*Id.* at 6.) Petitioner reported that she experienced a sudden change in vision in her right eye with images "appearing shattered" and brighter and more vibrant. (*Id.* at 8.) The episode lasted about twenty minutes and then resolved on its own. (*Id.*) Petitioner denied experiencing any migraines but reported frequent visual scotomas. (*Id.*) Petitioner was concerned for possible GCA. (*Id.*) PA Burns referred petitioner to an ophthalmologist for further evaluation. (*Id.* at 8-9.) PA Burns's referral order noted "hx PMR/GCA." (*Id.* at 17.)

Petitioner did not submit any records documenting her treatment beyond March 21, 2019.

#### **IV. Expert Opinions**

##### **a. Petitioner's Expert, Thomas Zizic, M.D.**

Petitioner presented an expert opinion from rheumatologist Thomas Zizic, M.D., in support of her claim. Dr. Zizic is a physician and Associate Professor of Medicine at Johns Hopkins Hospital and Johns Hopkins University School of Medicine in Baltimore, Maryland. (Ex. 10, p. 1; Ex. 11, p. 1.) He received his medical degree from Johns Hopkins University School of Medicine and completed an internship and residency in internal medicine at Johns Hopkins University Hospital Center. (Ex. 10, p. 1; Ex. 11, p. 1.) He later completed a post-doctoral fellowship in rheumatology at Johns Hopkins. (Ex. 10, p. 1.) From approximately 1973 to 1982, Dr. Zizic served as the Associate Director for the Rheumatic Disease Unit for Johns Hopkins at the Good Samaritan Hospital. (Ex. 10, p. 1; Ex. 11, p. 4.) He is a founding fellow of the American College of Rheumatology and previously served as President of the Maryland Society of Rheumatic Disease. (Ex. 10, pp. 1-2; Ex. 11, p. 3.) His research focuses on osteoarthritis, osteoporosis, and connective tissue diseases, including systemic lupus erythematosus, PMR, rheumatoid arthritis, and polymyositis. (Ex. 10, p. 2.) Additionally, Dr. Zizic has published approximately 100 articles and abstracts in peer-reviewed journals and various chapters in medical textbooks. (*Id.*; Ex. 11, pp. 7-18.)



Regarding petitioner's diagnosis, Dr. Zizic explained that PMR is an inflammatory condition that generally manifests in persons over fifty years of age. (Ex. 10, p. 20.) He stated that there are no laboratory tests used to diagnose the condition. (*Id.* at 21.) Instead, the diagnosis is made based on clinical presentation, inflammatory markers, and response to treatment. (*Id.*) While the cause of PMR is unknown, Dr. Zizic stated that it is widely understood in the medical community to be a cell-mediated autoimmune disease. (*Id.*) He elaborated that the disease manifests as an inflammatory reaction impacting the lining of a joint, often in the shoulders and hips, and occasionally the arteries. (*Id.*) Given that it is a cell-mediated autoimmune disease, Dr. Zizic asserted that patients with PMR are not expected to have autoantibodies. (Ex. 40, p. 3.)

Dr. Zizic agreed with petitioner's diagnosis of PMR but also suggested that petitioner also exhibited symptoms indicative of GCA, including neck pain and headache. (Ex. 40, p. 2.) However, Dr. Zizic acknowledged that petitioner "did not have sufficient clinical manifestations to diagnose giant cell arteritis." (*Id.* at 4.) Although petitioner was not diagnosed with GCA, Dr. Zizic opined that both diseases are types of vasculitis. (Ex. 10, p. 21.) He elaborated that PMR and GCA are "different manifestations of the same disease process." (*Id.* at 21-22; *see also* Ex. 40, p. 3 (discussing how PMR is on the mild spectrum of GCA).) Dr. Zizic emphasized that 40% of patients with GCA also have PMR, while 10% of PMR patients develop GCA in the course of their disease. (Ex. 10, p. 25; Ex. 40, p. 8.) He further noted that there is "evidence for the presence of identical vasculitis in both polymyalgia rheumatica and giant cell arteritis." (Ex. 40, p. 5 (citing D. Blockmans et al., *New Arguments for a Vasculitic Nature of Polymyalgia Rheumatica Using Positron Emission Tomography*, 38 RHEUMATOLOGY 444 (1999) (Ex. 14).) To Dr. Zizic, this suggests that like GCA, PMR is part of a vascular process. (*Id.* (citing Cornelia M. Weyand et al., *Tissue Cytokine Patterns in Patients with Polymyalgia Rheumatica and Giant Cell Arteritis*, 121 ANNALS INTERNAL MED. 484 (1994) (Ex. 36); Cornelia M. Weyand et al., *Disease Patterns and Tissue Cytokine Profiles in Giant Cell Arteritis*, 40 ARTHRITIS & RHEUMATISM 19 (1997) (Ex. 38).) Dr. Zizic added that it is significant that PMR is a type of vasculitis because dendritic cells in the adventitia-media border of the artery play a key role in causing vasculitis. (*Id.* at 4-5 (citing Cornelia M. Weyand & Jörg J. Goronzy, *Medium- and Large-Vessel Vasculitis*, 349 NEW ENGLAND J. MED. 160 (2003) (Ex. 41); Cornelia M. Weyand et al., *Vascular Dendritic Cells in Giant Cell Arteritis*, 1062 ANNALS NEW YORK ACAD. SCI. 195 (2005) (Ex. 37)).) Therefore, Dr. Zizic challenged Dr. Matloubian's assertion that there is no understanding of the pathogenesis of PMR. (*See id.* at 8 (reiterating that PMR and GCA "are at opposite ends of the severity spectrum of the same cell-mediated autoimmune vasculitis").)

Dr. Zizic offered molecular mimicry as a theory for causation. He opined that several environmental factors, including the flu vaccine, can "activate the immune system, leading to the development of antigen-specific T and B lymphocytes that can be activated as naïve cells or as memory cells." (Ex. 10, pp. 28-29.) He elaborated that the molecules contained in vaccines can cross-react with self-molecules and cause a cell-mediated autoimmune disease in genetically susceptible individuals. (*Id.* at 29-30.) Dr. Zizic relied on articles by Dessen et al. and Sun et al. to show that the "influenza

virus hemagglutinin 308-317 peptide shares a similar 3-dimensional structure with CII256-271 and can bind HLA-DR4/1 molecules with higher affinity.” (*Id.* at 29 (citing Andréa Dessen et al., *X-Ray Crystal Structure of HLA-DR4 (DRA\*0101, DRB1\*0401) Complexed with a Peptide from Human Collagen II*, 7 IMMUNITY 473 (1997) (Ex. 18); Jian Sun et al., *Superior Molecularly Altered Influenza Virus Hemagglutinin Peptide 308-317 Inhibits Collagen-Induced Arthritis by Inducing CD4+ Treg Cell Expansion*, 64 ARTHRITIS & RHEUMATISM 2158 (2012) (Ex. 32)).) Dr. Zizic further explained that identifying the specific self-antigen in petitioner is impossible due to the variety of different peptides recognized by autoreactive T cell receptors. He added that the antigens in cell-mediated immunity are in the inflamed tissue and are therefore often unidentifiable. (Ex. 40, pp. 8-9.) However, Dr. Zizic opined that both self-reactive T cells and B cells can result from epitope mimicry and cause autoimmune disease. (*Id.* at 9.) He noted that the Rose article cited by Dr. Matloubian acknowledges this fact. (See *id.* (citing Noel R. Rose, *Negative Selection, Epitope Mimicry and Autoimmunity*, 49 CURRENT OPINION IN IMMUNOLOGY 51 (2017) (Ex. A, Tab 12)).) Dr. Zizic also opined that in addition to the flu vaccine petitioner received, other factors, such as history of earlier flu vaccines and effects of aging, can predispose an individual to autoimmunity and cause the breakdown of tolerance to self via molecular mimicry. (Ex. 10, pp. 27-29; Ex. 40, pp. 6-8.)

Dr. Zizic offered several case reports to support his opinion, noting that forms of vasculitis, including PMR and GCA, have been reported after the flu vaccination. (Ex. 10, pp. 19-20 (citing Rainer Birck et al., *ANCA-Associated Vasculitis Following Influenza Vaccination*, 15 J. CLINICAL RHEUMATOLOGY 289 (2009) (Ex. 12); Caterina Bonetto et al., *Vasculitis as an Adverse Event Following Immunization – Systematic Literature Review*, 34 VACCINE 6641 (2016) (Ex. 15); Eric Liozon et al., *Polymyalgia Rheumatica Following Influenza Vaccination*, 48 J. AM. GERIATRICS SOC’Y 1553 (2000) (Ex. 25); Juan Marti & Enrique Anton, *Polymyalgia Rheumatica Complicating Influenza Vaccination*, 52 J. AM. GERIATRICS SOC’Y 1412 (2004) (Ex. 26); Carlos Perez & Elias Maravi, *Polymyalgia Rheumatica Following Influenza Vaccination*, 23 MUSCLE & NERVE 824 (2000) (Ex. 27); A. Soriano et al., *Giant Cell Arteritis and Polymyalgia Rheumatica After Influenza Vaccination: Report of 10 Cases and Review of the Literature*, 21 LUPUS 153 (2012) (Ex. 31); Makoto Wada et al., *Giant Cell Arteritis with Polymyalgia Rheumatica Associated with Influenza Vaccination*, 38 J. DERMATOLOGY 1099 (2011) (Ex. 33)).)

While Dr. Zizic did not extensively address the issue of timing in his reports, he noted that petitioner first experienced symptoms of PMR two weeks after receiving the flu vaccine. (See Ex. 40, p. 1.) Additionally, Dr. Zizic offered a case report that found that a range of 12 to 21 days was an appropriate temporal relationship for onset of vasculitis following the flu vaccine. (Yaron Zafirir, Nancy Agmon-Levin, & Yehuda Shoenfeld, *Post-Influenza Vasculitides: A Possible New Entity*, 15 J. CLINICAL RHEUMATOLOGY 269, 270 (2009) (Ex. 39)).



**b. Respondent's Expert, Mehrdad Matloubian, M.D., Ph.D.**

Respondent offered an expert opinion from immunologist and rheumatologist Mehrdad Matloubian, M.D., Ph.D. Dr. Matloubian is board-certified in rheumatology and has a Ph.D. in virology and immunology. (Ex. A, p. 1; Ex. B, pp. 1-2.) He is currently a physician and Associate Professor of Medicine in the rheumatology division at the University of California, San Francisco. (Ex. A, p. 1; Ex. B, p. 2.) He has expertise in T and B cell responses to viruses and factors that regulate lymphocyte circulation and trafficking. (Ex. A, p. 1.) His research primarily focuses on innate and adaptive immune responses of T and B cells to acute and chronic viral infections. (*Id.*) Additionally, Dr. Matloubian has published several peer-reviewed articles on these topics. (*Id.*; Ex. B, pp. 10-14.)

Dr. Matloubian explained that PMR is an inflammatory disease of unknown etiology. (Ex. A, p. 3.) He noted that symptoms of PMR include aching and morning stiffness in the shoulders, hip girdle, and neck. (*Id.*) Given that MRI or ultrasound imaging have shown bursitis or synovitis in patients with PMR, Dr. Matloubian explained that PMR is understood to be a disease of the joints underlying the muscle groups. (*Id.* at 3-4.) He discussed that it is usually a systemic inflammatory disease characterized by elevated inflammatory markers such as ESR and/or CRP. (*Id.*) He further stated that there is no known trigger for the disease. (*Id.*) Dr. Matloubian opined that petitioner's course of PMR was typical for the disease given her occasional flares and responsiveness to prednisone. (*Id.* at 4.) He added that it is not unusual for a previously healthy person to experience an abrupt onset of PMR as petitioner experienced. (*Id.* at 3; Ex. C, p. 3.)

In response to Dr. Zizic's discussion of the similarities between PMR and GCA, Dr. Matloubian noted that while petitioner had some neck symptoms and a headache, petitioner did not have any other symptoms suggestive of GCA and was never diagnosed with the disease. (Ex. A, p. 4.) He also discussed some differences between the two diseases. He noted that GCA often requires treatment with higher doses of prednisone than PMR. (*Id.*) While petitioner was treated with 5-20 mg of prednisone daily throughout the course of her disease, Dr. Matloubian noted that treatment of GCA requires prednisone in the 40-60 mg per day range. (*Id.*) He added that GCA is diagnosed based on a temporal artery biopsy, which petitioner never had done. (Ex. C, p. 2.) Dr. Matloubian elaborated that there was insufficient evidence in the medical records to diagnose petitioner with GCA. (*Id.*) Therefore, Dr. Matloubian concluded that it was unlikely petitioner had GCA.

Dr. Matloubian challenged molecular mimicry as a plausible mechanism by which the flu vaccine can cause PMR. Given that the medical community's understanding of PMR's pathogenesis is "primitive" and its mechanism is unknown, Dr. Matloubian opined that "the sequence of events that leads to development of inflammation and secretion of inflammatory cytokines, such as IL-6, is not known." (Ex. A, p. 4.) He added that the "absence of autoantibodies in PMR to a specific component of the musculoskeletal system suggests that this disease may not be antigen-specific, and

most likely not induced through processes such as molecular mimicry.” (*Id.*) Additionally, since most PMR patients quickly respond to low doses of prednisone, Dr. Matloubian explained that this suggests that unlike other autoimmune diseases such as rheumatoid arthritis, PMR is not antigen-driven or characterized by persistent inflammation. (*Id.*) He further explained that that “no foreign or self-antigens have been identified as the target of the immune response that leads to the development of PMR.” (*Id.* at 6; see also Ex. C, p. 3.) Given that PMR primarily affects the elderly population, Dr. Matloubian opined that PMR is most likely caused by aging immune systems and “change[s] in balance towards pro-inflammatory pathways.” (Ex. A, pp. 5-6.) He concluded that without “an understanding of the basic pathogenesis of PMR, any medical theory set forth linking [the] influenza vaccination and development of PMR is purely speculative and unreliable.” (*Id.* at 6.)

Additionally, for molecular mimicry to be a plausible mechanism by which the flu vaccine can cause a disease, Dr. Matloubian explained that the flu infection should also be capable of triggering the disease. (Ex. A, p. 9; Ex. C, p. 2 (citing Rose, *supra*, at Ex. A, Tab 12).) However, as noted by Dr. Matloubian, researchers have not identified an infectious cause for PMR. (Ex. A, p. 10; Ex. C, pp. 2-3.) Dr. Matloubian cited data from the World Health Organization, which showed no higher incidence of PMR following influenza outbreaks. (Ex. A, p. 10.) Therefore, Dr. Matloubian opined that molecular mimicry is an unlikely mechanism by which the flu vaccine could cause PMR. (*Id.*)

Dr. Matloubian identified several flaws in Dr. Zizic’s opinion. He asserted that Dr. Zizic’s theory is unsound because although Dr. Zizic identified collagen as a self-antigen in PMR, collagen is a T cell antigen associated with rheumatoid arthritis, type I diabetes, and multiple sclerosis, but has not been recognized as a self-antigen in PMR. (Ex. A, p. 10; Ex. C, p. 3.) Further, Dr. Matloubian challenged the literature Dr. Zizic relied on to support his theory that the hemagglutinin contained in the flu vaccine cross-reacts with collagen to trigger PMR. Regarding the Dessen et al. article (Ex. 18), Dr. Matloubian noted that the authors do not discuss molecular mimicry or recognize hemagglutinin and collagen as structurally similar. (Ex. A, p. 11.) Additionally, Dr. Matloubian noted that the Sun et al. article (Ex. 32) merely shows that an altered hemagglutinin peptide can inhibit T cell responses to a collagen-derived peptide in mice and rheumatoid arthritis patients. (*Id.* at 13.) However, the Sun et al. authors do not discuss molecular mimicry. (*Id.*)

Further, Dr. Matloubian disputed the relevance of the case reports Dr. Zizic relied on to bolster his theory. In the Liozon et al. case report, the subject’s symptoms appeared two days after receiving the flu vaccination; however, she also had a urinary tract infection requiring intravenous steroids. (Liozon et al., *supra*, at Ex. 25.) Dr. Matloubian explained that the patient’s urinary tract infection complicated any alleged vaccine association. (Ex. A, p. 7.) Additionally, in the Soriano et al. and Wada et al. case reports, which both explore reports of PMR and/or GCA, the subjects’ onset of symptoms ranged from one day to three months post-vaccination. (Soriano et al., *supra*, at Ex. 31; Wada et al., *supra*, at Ex. 33.) Dr. Matloubian opined, “[s]uch a large difference in the timing of onset of symptoms is not compatible with a unifying

immunological mechanism and is more suggestive of a coincidental rather than a causal association between vaccination and disease.” (Ex. A, p. 7.) Regarding the Soriano et al. case report, Dr. Matloubian noted that many of the vaccines examined contained adjuvants; conversely, the vaccine petitioner received did not contain adjuvants. (*Id.*) Dr. Matloubian also explained that several of the case reports Dr. Zizic referenced examined types of vasculitis associated with antineutrophil cytoplasmic antibodies (“ANCA”) and are therefore irrelevant to the case as petitioner did not suffer an ANCA-associated disease. (*Id.* (citing Birck et al., *supra*, at Ex. 12; Bonetto et al., *supra*, at Ex. 15; Zafrir, Agmon-Levin, & Shoenfeld, *supra*, at Ex. 39).) Dr. Matloubian also asserted that the case reports show only a temporal association between vaccination and disease but do not support causality. (*Id.*)

Regarding timing, Dr. Matloubian agreed that petitioner’s PMR symptoms “became apparent” after receiving her flu vaccine. (Ex. A, p. 6.) However, Dr. Matloubian appeared to suggest that petitioner’s PMR may have been dormant before it manifested clinically. (*Id.*) He explained that autoimmune diseases are often “years in the making and only diagnosed when they become clinically apparent.” (*Id.*) He therefore cautioned against inferring causation due to a temporal association between vaccination and clinical manifestation of symptoms. (See *id.* at 5-6.) Given Dr. Matloubian’s opinion that the pathogenesis of PMR is unknown, Dr. Matloubian suggested that there is no scientifically appropriate timeframe for which to infer vaccine-causation. (*Id.*)

## V. Discussion

As explained above, petitioner’s burden is to demonstrate by preponderant evidence, each of the three *Althen* prongs used to determine actual causation (i.e., an acceptable medical theory, a logical sequence of cause and effect, and a proximate temporal relationship). *Althen*, 418 F.3d at 1278.

### a. *Althen* Prong One

Under *Althen* prong one, petitioner must provide a “reputable medical theory,” demonstrating that the vaccine received can cause the type of injury alleged. *Pafford v. Sec’y of Health & Human Servs.*, 451 F.3d 1352, 1355-56 (Fed. Cir. 2006) (citations omitted). Such a theory must only be “legally probable, not medically or scientifically certain.” *Knudsen*, 35 F.3d at 549. Petitioner may satisfy the first *Althen* prong without resort to medical literature, epidemiological studies, demonstration of a specific mechanism, or a generally accepted medical theory. *Andreu v. Sec’y of Health & Human Servs.*, 569 F.3d 1367, 1378-79 (Fed. Cir. 2009) (citing *Capizzano v. Sec’y of Health & Human Servs.*, 440 F.3d 1317, 1325-26 (Fed. Cir. 2006)). However, “[a] petitioner must provide a ‘reputable medical or scientific explanation’ for [her] theory. While it does not require medical or scientific certainty, it must still be ‘sound and reliable.’” *Boatmon*, 941 F.3d at 1359 (quoting *Knudsen*, 35 F.3d at 548-49).

Dr. Zizic summarizes his theory of causation as follows:

Inflammation and damage involving the walls of blood vessels in the vascular syndromes have been attributed to immunologic mechanisms. Although immune-complex mediated mechanisms appear to be operative in some vasculitides, cellular immune mechanisms are more likely to be important in the initiation and perpetuation of lesions in patients with polymyalgia rheumatica and giant cell arteritis. As discussed above, in genetically susceptible individuals, influenza antigens in the vaccine lead to cross-reactivity and molecular mimicry to self-antigens that then break tolerance to self. Some individuals develop humoral autoimmune diseases such as rheumatoid arthritis and others develop cell-mediated autoimmune diseases such as polymyalgia rheumatica and giant cell arteritis.

(Ex. 10, p. 30.)

Petitioner's causation theory has several shortcomings. First, Dr. Zizic opined that the hemagglutinin contained in the flu vaccine can cross react with type II collagen, thus triggering activation of B and/or T lymphocytes through molecular mimicry based largely upon articles by Dessen et al. and Sun et al. (Ex. 10, p. 29 (citing Dessen et al., *supra*, at Ex. 18; Sun et al., *supra*, at Ex. 32).) However, Dr. Matloubian explained that neither the Dessen et al. article nor the Sun et al. article discussed molecular mimicry or identified hemagglutinin and collagen as structurally similar. (Ex. A, pp. 11-13.) Further, Dr. Matloubian persuasively explained that while collagen is a self-antigen associated with rheumatoid arthritis, it had not been identified as a self-antigen in PMR. (*Id.* at 10.) In fact, Dr. Matloubian asserted that "no foreign or self-antigens have been identified as the target of the immune response that leads to the development of PMR." (*Id.* at 6.) Dr. Matloubian further opined that for molecular mimicry to be a viable mechanism by which the flu vaccine can trigger PMR, the flu infection must be capable of causing the disease. (*Id.* at 9.) However, since PMR is a disease of unknown cause, an infectious etiology has not been identified, and neither the flu vaccine nor the flu infection has been associated with the disease. (*Id.* at 9-10.) In fact, Dr. Matloubian indicates that no trigger of any kind has been identified in the development of PMR.<sup>4</sup> (*Id.* at 4.)

In response to Dr. Matloubian's criticism, Dr. Zizic acknowledged that self-antigens specific to PMR are unidentifiable. (See Ex. 40, pp. 8-9.) However, Dr. Zizic was critical of Dr. Matloubian's suggestion that identifiable autoantibodies are necessary, stressing instead that the condition at issue is a cell-mediated autoimmune disease. (*Id.* at 3.) In that regard, Dr. Zizic stresses a hypothesis of GCA development, contending that GCA and PMR are "different manifestations of the same disease

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<sup>4</sup> In a previous Vaccine Program case, a special master rejected molecular mimicry as a mechanism by which the flu vaccine can cause PMR. *C.P. v. Sec'y of Health & Human Servs.*, No. 14-917V, 2019 WL 5483621, at \*28 (Fed. Cl. Spec. Mstr. Aug. 21, 2019). In *C.P.*, the special master found it significant that there are no autoantibodies or identifiable antigens in patients with PMR and that as a result, no sequence of amino acids can be compared to those in the flu vaccine. *Id.* at \*26, 28. The special master in *C.P.* also credited Dr. Matloubian's opinion that the pathogenesis of PMR is unknown and there is no established association between the flu virus or vaccine and PMR. *Id.* at \*28.

process.” (Ex. 10, p. 22; Ex. 40, p. 7.) Dr. Zizic suggests that a number of reports have linked PMR to the flu vaccine in connection with GCA. (Ex. 10, p. 20.) He further suggests that GCA is “considered by many people to be the best example of a TH1 vasculitis.” (*Id.* at 21.)

Specifically, he quotes the following:

The cause of GCA is unknown, but the vessel wall inflammation is believed to be predominantly cell-mediated, rather than autoantibody-induced. Dendritic cells residing in the adventitia become activated by an unknown antigen, and signal for T lymphocytes to enter the vessel wall via the vasa vasorum. Activated T cells differentiate and clonally expand, producing INF-gamma, which results in macrophage infiltration. Macrophages infiltrate all layers of the arterial wall, secreting a broad range of inflammatory cytokines . . . .

(Ex. 40, p. 7.) However, even if GCA and PMR have some relation,<sup>5</sup> application of this particular theory to PMR is unpersuasive.

One study cited by Dr. Zizic reviewed patients with either GCA or PMR who were tested by temporal artery biopsy. (Weyand et al., *supra*, at Ex. 36, p. 484.) While the study found similarities in inflammatory cytokine profiles (most notably IL-2) between GCA and PMR patients, the biopsy results showed that none of the subjects experiencing PMR alone had evidence of arterial inflammatory infiltrate.<sup>6</sup> (*Id.* at 487.) The authors hypothesized that PMR may include subclinical vascular involvement, but also noted that there may be important differences between PMR and GCA patients. In particular, they proposed that INF-gamma, which was not found among PMR patients, may be essential for the development of vasculitis. (*Id.* at 489-90.) A follow up study further explored the relationship between inflammatory cytokines and disease expression among GCA patients. (Weyand et al., *supra*, at Ex. 38.) That study indicated that INF-gamma is likely “a critical cytokine in GCA” given its documented

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<sup>5</sup> The comorbidity of PMR and GCA and the fact that they are both age-related inflammatory conditions has led some to propose that PMR may also be a vasculitis and that the two conditions may exist on a spectrum. (*E.g.*, Blockmans et al., *supra*, at Ex. 14; Weyand et al., *supra*, at Ex. 36.) Despite this hypothesis, researchers have acknowledged that the relationship between the two syndromes remains “unclear.” (Weyand et al., *supra*, at Ex. 38, p. 20.) In fact, only 15% of PMR patients later develop vasculitis. (Weyand et al., *supra*, at Ex. 36, p. 490.) Conversely, “only a fraction” of GCA patients develop symptoms characteristic of PMR. (Weyand et al., *supra*, at Ex. 38, p. 24.) For his part, Dr. Matloubian does not dispute that there is overlap between GCA and PMR or that PMR may involve subclinical vascular inflammation, but stresses that the cause of neither condition is known, let alone having any causal relationship related to influenza antigen. (Ex. C, p. 2.) Dr. Zizic likewise acknowledges that ultimately the cause of GCA remains unknown. (Ex. 40, p. 7.) In another case involving PMR (relative to the Tdap vaccine), the same special master presiding in *C.P.* discussed the comparison between PMR and GCA in more detail. See *Suliman v. Sec’y of Health & Human Servs.*, No. 13-993V, 2018 WL 6803697, at \*25, 28 (Fed. Cl. Spec. Mstr. Nov. 27, 2018). In *Suliman*, the special master emphasized that PMR is distinguishable from GCA, and that the petitioner did not have GCA. *Id.*

<sup>6</sup> Dr. Zizic suggests that the temporal artery biopsy is not dispositive of whether vasculitis exists (Ex. 10, pp 22-23), though he does agree that it is the “gold standard” for detecting GCA (*Id.* at 21).



absence from patients without overt vasculitis. (*Id.* at 24.) Citing back to the prior study, the authors explained that while the IL-2 demonstrated among PMR patients and the INF-gamma seen among GCA patients may both be secreted by Th1 helper cells, “several lines of evidence indicate that production of both cytokines is not necessarily linked.” (*Id.* at 25.) Further, they observed that in the follow-up study there was no correlation between IL-2 and INF-gamma in individual patients. (*Id.*) Despite acknowledging this distinction (Ex. 10, p. 24), Dr. Zizic specifically invokes INF-gamma production as part of his explanation of GCA as a cell-mediated autoimmune process (*Id.* at 25). Yet, extension of that mechanism to PMR does not appear to be reliable based on the above-discussed literature. In that regard, Dr. Matloubian emphasized that because the medical community’s understanding of PMR’s pathogenesis is “primitive,” it is impossible to identify “the sequence of events that leads to development of inflammation and secretion of inflammatory cytokines.” (Ex. A, p. 4.)

The case reports Dr. Zizic relied on cannot remedy the deficiencies in his theory. While case reports are not wholly without evidentiary value, the case reports show only a temporal association and do not establish causality. (See Ex. A, p. 7.) Additionally, many of the case reports Dr. Zizic relied on document ANCA-associated vasculitis following the flu vaccine, further diminishing their value. (See Birck et al., *supra*, at Ex. 12; Bonetto et al., *supra*, at Ex. 15; Zafirir, Agmon-Levin, & Shoenfeld, *supra*, at Ex. 39.) Dr. Matloubian opines that the ANCA-associated vasculitis case reports are entirely irrelevant, because it is a different condition and petitioner was never diagnosed as having it. (Ex. A, p. 7.)

A petitioner must provide a “‘reputable medical or scientific explanation’ for [her] theory.” *Boatmon*, 941 F.3d at 1359 (quoting *Knudsen*, 35 F.3d at 548-49). Here, Dr. Zizic was unpersuasive in either seeking to identify components of the flu vaccine that could be mimicked to cause PMR or in alternatively invoking GCA to circumstantially identify a mechanism of cell-mediated autoimmunity. And, although petitioner is not required to prove an exact biological mechanism, she has not otherwise provided any sound and reliable theory of causation. Therefore, petitioner has failed to meet her burden to show that the flu vaccine can cause PMR.

#### **b. *Althen* Prong Two**

The second *Althen* prong requires proof of a logical sequence of cause and effect, usually supported by facts derived from a petitioner’s medical records. *Althen*, 418 F.3d at 1278; *Andreu*, 569 F.3d at 1375-77; *Capizzano*, 440 F.3d at 1326; *Grant*, 956 F.2d at 1148. In establishing that a vaccine “did cause” injury, the opinions and views of the injured party’s treating physicians are entitled to some weight. *Andreu*, 569 F.3d at 1367; *Capizzano*, 440 F.3d at 1326 (quoting *Althen*, 418 F.3d at 1280) (stating that “medical records and medical opinion testimony are favored in vaccine cases, as treating physicians are likely to be in the best position to determine whether a ‘logical sequence of cause and effect show[s] that the vaccination was the reason for the injury’”). However, medical records and/or statements of a treating physician’s views do not *per se* bind the special master to adopt the conclusions of such an individual, even if



they must be considered and carefully evaluated. See Section 13(b)(1) (providing that “[a]ny such diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court”); *Snyder v. Sec’y of Health & Human Servs.*, 88 Fed. Cl. 706, 746 n.67 (2009) (stating that “there is nothing . . . that mandates that the testimony of a treating physician is sacrosanct—that it must be accepted in its entirety and cannot be rebutted”).

To support a logical sequence of cause and effect, petitioner primarily relies on statements from Dr. Bransdorf and PA Burns. Petitioner argues that both Dr. Bransdorf and PA Burns noted that petitioner’s symptoms of joint pain two weeks post-vaccination may have been due to her flu vaccine. (See ECF No. 50, p. 17; ECF No. 55, p. 6.) However, these statements do not carry petitioner’s burden under *Althen* prong two. PA Burns suggested that petitioner was having a “flu like” reaction to the vaccine (*id.* at 6); however, petitioner was not diagnosed with PMR until several weeks later and neither PA Burns nor Dr. Bransdorf ever attributed petitioner’s PMR to the flu vaccine. Nor, in any event, do these statements bind a special master to adopt their conclusions. *Snyder*, 88 Fed. Cl. at 746, n.67. Further, in light of my finding that petitioner has not satisfied *Althen* prong one, the statements from her treating physicians carry little weight. See *Langland v. Sec’y of Health & Human Servs.*, 109 Fed. Cl. 421, 438 (2013) (stating that “in the absence of a theory showing a vaccine *could* cause a particular injury, a doctor’s belief that it *did* will not prove causation) (emphasis in original); see e.g., *Deshler v. Sec’y of Health & Human Servs.*, No. 16-1070V, 2020 WL 4593162, at \*21 (Fed. Cl. Spec. Mstr. July 1, 2020).

Although Dr. Zizic suggested that the temporal association between petitioner’s flu vaccine and onset of PMR indicated a logical sequence of cause and effect, both Dr. Zizic and Dr. Matloubian agreed that onset of PMR is typical in individuals over fifty years of age. (Ex. 10, p. 20; Ex. A, p. 5.) Because PMR generally occurs in the elderly population, Dr. Matloubian indicated that the disease is likely the result of an aging immune system. (Ex. A, p. 5.) Though generally active and healthy, petitioner was 73 years old at the time of vaccination. (Ex. 1, p. 1; Ex. 3, p. 7.) Further, Dr. Matloubian asserted that PMR can be dormant for years before individuals experience clinical manifestations. (Ex. A, p. 6.) Thus, the temporal association between petitioner’s flu vaccine appears to be merely coincidental. Additionally, temporal association alone cannot satisfy a petitioner’s burden under *Althen* prong two. See *C.P.*, 2019 WL 5483621, at \*29 (citing *Grant*, 956 F.2d at 1148).

Dr. Zizic also attempted to show that the flu vaccine can cause PMR based on PMR’s similarities to GCA. However, as Dr. Zizic acknowledged, GCA accompanies only a subset of PMR cases. (Ex. 10, p. 25; Ex. 40, p. 8.) Further, although petitioner expressed concern for GCA (Ex. 5, p. 16; Ex. 42, p. 8), neither Dr. Bransdorf nor PA Burns ever diagnosed petitioner with GCA. As discussed above, Dr. Matloubian’s opinion that PMR is a disease of unknown etiology is persuasive. Thus, Dr. Zizic’s suggestion that petitioner’s PMR indicates vaccine-causation based on a GCA comorbidity does not establish a logical sequence of cause and effect. The evidence supports a finding that petitioner’s PMR was idiopathic rather than vaccine induced.

Accordingly, petitioner has failed to satisfy *Althen* prong two.

### c. *Althen* Prong Three

The third *Althen* prong requires establishing a “proximate temporal relationship” between the vaccination and the injury alleged. *Althen*, 418 F.3d at 1281. That term has been equated to the phrase “medically-acceptable temporal relationship.” *Id.* A petitioner must offer “preponderant proof that the onset of symptoms occurred within a timeframe which, given the medical understanding of the disorder’s etiology, it is medically acceptable to infer causation.” *de Bazan v. Sec’y of Health & Human Servs.*, 539 F.3d 1347, 1352 (Fed. Cir. 2008). The explanation for what is a medically acceptable timeframe must coincide with the theory of how the relevant vaccine can cause an injury (*Althen* prong one’s requirement). *Id.* at 1352; *Shapiro v. Sec’y of Health & Human Servs.*, 101 Fed. Cl. 532, 542 (2011), *recons. den’d after remand*, 105 Fed. Cl. 353 (2012), *aff’d mem.*, 503 Fed. Appx. 952 (Fed. Cir. 2013); *Koehn v. Sec’y of Health & Human Servs.*, No. 11-355V, 2013 WL 3214877 (Fed. Cl. Spec. Mstr. May 30, 2013), *mot. for review den’d* (Fed. Cl. Dec. 3, 2013), *aff’d*, 773 F.3d 1239 (Fed. Cir. 2014).

The parties do not dispute that petitioner manifested symptoms of PMR two weeks after receiving the flu vaccine; however, Dr. Matloubian persuasively explained that because petitioner has not shown that the flu vaccine can cause PMR, there is no appropriate temporal association. (See Ex. A, pp. 5-6.) Thus, petitioner cannot satisfy her burden under *Althen* prong three. See *Langland*, 109 Fed. Cl. at 443 (“[T]o satisfy the ‘proximate temporal relationship’ prong of the *Althen* test, petitioners must demonstrate, by a preponderance of the evidence, that the onset of symptoms occurred within a time frame for which it is medically acceptable to infer causation-in-fact . . . With no reputable theory as to how the vaccination could cause the injury, this exercise is not possible.”) (citing *de Bazan*, 539 F.3d at 1352); see also *Suliman v. Sec’y of Health & Human Servs.*, No. 13-993V, 2018 WL 6803697, at \*30 (Fed. Cl. Spec. Mstr. Nov. 27, 2018) (finding that petitioner could not satisfy burden under *Althen* prong three because petitioner failed to show that the Tdap vaccine could cause PMR and/or myositis.)

## VI. Conclusion

Accordingly, for all the reasons described above, petitioner is not entitled to compensation for her PMR. Therefore, this case is dismissed.<sup>7</sup>

**IT IS SO ORDERED.**

**s/Daniel T. Horner**

Daniel T. Horner  
Special Master

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<sup>7</sup> In the absence of a timely-filed motion for review of this Decision, the Clerk of the Court shall enter judgment accordingly.